5. **(Amended)** The method of claim 4, wherein the inhibitor decreases expression of a gene encoding ERK1/2, a MEK and/or a JNK.

6. **(Amended)** The method of claim 5, wherein the inhibitor interacts with an ERK1/2, a MEK and/or JNK gene.

8. **(Amended)** The method of claim 3, wherein the inhibitor decreases the activity of ERK1/2, a MEK and/or a JNK.

9. **(Amended)** The method of claim 6, wherein the inhibitor interacts with ERK1/2, a MEK and/or a JNK protein.

10. **(Amended)** The method of claim 6, wherein the inhibitor inhibits ERK1/2, a MEK and/or JNK phosphorylation.

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Please insert new claims 19-28:

19. **(New)** The method of claim 1, wherein the inhibitor is a dominant negative mutant of ERK1/2, a MEK and/or a JNK.

20. **(New)** The method of claim 1, wherein the subject is overweight or obese.

21. **(New)** The method of claim 1, wherein the disease or condition is caused, or contributed to, by TNF-α induced lipolysis.

22. **(New)** The method of claim 1, wherein the disease or condition is caused, or contributed to, by basal lipolysis.

23. **(New)** The method of claim 22, wherein the inhibitor does not interact with a PPAR-γ receptor and the inhibitor is not sodium salicylate.

24. **(New)** The method of claim 23, wherein the inhibitor is selected from the group consisting of an antisense molecule, a triplex molecule, a ribozyme and a dominant negative mutant targeted to ERK1/2 or a MEK.

25. **(New)** The method of claim 1, further comprising determining the level of activity of ERK1/2 or a MEK in the subject.

26. **(New)** The method of claim 25, wherein the level of activity of ERK1/2 or a MEK is determined in a sample of fat cells from the subject.

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27. **(New)** The method of claim 1, wherein the inhibitor is administered in the presence of a carrier that facilitates entry of the inhibitor into cells of the subject.

28. **(New)** The method of claim 1, wherein the inhibitor is administered locally.